ANATOMICAL FINDINGS IN TWO CASES OF KORSAKOFF'S SYMPTOM-COMPLEX.

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In 1887 Korsakoff published his first descriptions of mental disturbance associated with multiple neuritis. Since then much has been written about this symptom-complex; but comparatively few cases with anatomical findings have been reported.

The following cases, which occurred in the medical service of the Boston City Hospital, are apparently of alcoholic origin and died in an early stage of the disease.

The first case is an example of an acute polyneuritis with a chronic mild delirium, developing symptoms resembling cerebral hemorrhage. The second case is more chronic in onset and more typical of Korsakoff's symptom-complex. Both exhibit marked changes in many parts of the nervous system.

I wish here to express my obligations to Dr. J. W. Bartol and H. D. Arnold for the use of their clinical records. The autopsies were performed at the Boston City Hospital during the pathological service of Dr. F. B. Mallory. I wish also to thank Dr. E. E. Southard and Dr. A. M. Barrett for suggestions as to the anatomical interpretation.

CASE I. (FROM SERVICE OF DR. J. W. BARTOL.)

S. S., a woman, age forty-eight, married, cook, was admitted to the Boston City Hospital, September 10, 1903. Family history was negative. Patient had no children. There were two miscarriages, no cause known. She had slow fever and measles as a child, also asthma. For several years she had been addicted to liquor, and for the last few months had taken two or three glasses of whiskey daily. She considered herself well up to two weeks previous to admission. At that time she commenced to have pains in her knees and ankles, and later she became unable to walk. Her legs were often swollen at night. She was a well-developed and obese person. The pupils reacted promptly to light and in accommodation. Tongue was clean. There was a systolic murmur of the heart, the pulmonic second being accentuated. Lungs were negative. In the extremities there was no swelling or tenderness of the joints, but some tenderness of the muscles. Knee-jerks were absent.

Sept. 14.—Some delirium at night. She was talking considerably, frequently attempting to get out of bed. There was marked wrist-drop, with inability to use the fingers.

Sept. 22.—Temperature remained normal. Patient was sleeping fairly well. Frequently woke up and called for various friends, thinking that she saw them. Complete toe-drop and wrist-drop.

Sept. 24.—There was noted a convulsion with twitching of the face, neck, and shoulders of both sides; more prominent on the left. This convulsion lasted about 45 minutes. She was unconscious and could not be aroused. After this the mental condition was the same as prior to the attack. She remained quiet the rest of the day and night. On the following day there was another convulsion, in which both sides of the face, abdominal muscles, and the legs to a slight extent, were affected.

There was spasticity of the right arm. She was unconscious with incontinence. This attack lasted about four hours; was quieted with morphine.

Sept. 26—She was in a stuporous condition from which she could be fairly easily aroused. She recognized her name and contradicted the physician on being called by her wrong name. During the afternoon and night, when disturbed there were noted explosions or clonic spasms of the face, and to a less degree of the body. The labio-nasal fold was indistinct and there was a decided droop to the left side of the face. Tongue was protruded to the left. There was flaccidity of the left arm and leg. She seemed to recognize her husband. She kept up considerable talking prior to the convulsion. Pupils were small, irregular in outline, and reacted to light. There was deviation to the right of both eyes. Pulse and temperature commenced to rise.

Sept. 30.—Mental condition was much the same. There were times when she was more sane and made clever replies to questions. She showed a tendency to fabricate, frequently speaking of incidents which probably had occurred in her past life. Respirations were normal. Pulse was 160. Temperature had risen to 104.5°. She began to vomit and was unable to retain any nourishment. Mental condition gradually grew worse. She rapidly became more feeble, and died Oct. 1, 1903.

Clinical Summary.—Acute alcoholic multiple neuritis in a woman of forty-eight. Accompanied by delirium, hallucinosis, and romancing. Later convulsions with twitchings of various muscle groups, which was followed by spasticity of one extremity and flaccidity of the extremities of the opposite side. Some paralysis of the facial muscles. Death after five weeks, preceded by a rapid rise in pulse and temperature.

Anatomical Findings.—Autopsy five hours post-mortem (Drs. R. E. Lee and E. E. Southard).

Well developed, obese white woman.

Peritoneal Cavity—Mesenteric lymph nodes normal.

Pleural Cavity—Normal.

Heart—Weight, 330 gms. Subepicardial fat increased in amount. Myocardium normal in color and firm. Endocardium normal. Mitral valve slightly thickened at the edges. Slight amount of sclerosis seen back of the cusps of the aortic valve.

Measurements—Tricuspid valve 11 cm.; pulmonary valve 7 cm.; mitral valve 11 cm.; aortic valve 7 cm.; left ventricle 1.75 cm.; right ventricle 3.24 cm.

Fresh specimen examined with acetic acid shows no fat.

Lungs—Left dark red and edematous. Posterior part of the upper lobe is firm and contains little air. Cut surfaces granular. Small greenish bronchial plugs of pus exude on slight pressure.

Right Lung—Dark red and moist. Posterior part shows the same process as in the left lung, but not so marked.

Spleen—Weight, 55 gms. Capsule ruptures easily. Trabeculæ not increased. Malpighian bodies seen with difficulty.

Gastrointestinal Tract—Normal.

Liver—Weight, 1760 gms. Rather yellow in color, greasy to the feel. Central veins injected. Surface of the liver slightly roughened and adherent at several points to diaphragm and adjacent viscera.

Kidneys—Weight, 210 gms. Normal. Fresh specimen examined with acetic acid shows no fat.

Brain—Weight, 1,100 gms.

Head—Scalp and calvarium normal. Dura everywhere free and tense. Brain appears to retract slightly on removal of skull-cap. Arachnoidal villi moderately developed. Pia almost dry. Vessels normal. Veins of sulci moderately injected. Substance normal. Ventricles contain a normal amount of fluid. Inter-brain, mid-brain, and pontine region normal on section. The medulla cuts well, except in ill-defined fusiform area of slightly reduced consistence and slightly pinker tinge to the left of raphe in the formatio reticularis a few mm. dorsal to the olive. The left side of the medulla is slightly larger than the right and the olive seems pushed ventrally by 1-2 mm. Remainder of bulb and cord normal.

Microscopical Findings.—Lungs—Marked congestion in places with more or less hemorrhage. Some foci of exudation containing polynuclear leucocytes, serum, fibrin, and red blood corpuscles. Pigmented mononuclears are fairly abundant in places. A variety of organisms are present.

Spleen—Not notable except for much brownish pigment in coarse granules within phagocytic cells.

Liver—Shows marked fatty infiltration which is least abundant adjoining the portal vessels. There is some increase of portal connective tissue with formation of so-called bile ducts.

Kidney—Shows atrophyof numerous glomeruli and tubules and increase of connective tissue. Moderate congestion of the vessels in the pyramids.

Thyroid—Many of the glands are dilated and filled with colloid material. There is some increase of connective tissue. Many foci of lymphoid and plasma cells.

Peripheral Nerves—With Marchi method intense degeneration of internal and external popliteal, the ulnar and musculospiral nerves of both sides. Both anterior crural nerves and median nerves (at the elbow) show many degenerated fibers, but not so marked as the above. The brachial plexuses have a few affected fibers, more marked on the right side.

The vagi and phrenic nerves present numerous degenerated fibers. Other cranial nerves were not examined.

Cord—Anterior horn cells of the lumbar, sacral and cervical enlargements show typical acute changes of the axonal type. There is considerable excess of yellowish granular pigment arranged peripherally, usually occupying the basal portion.

The lumbar and sacral segments are most severely involved. Most of the cells of the lateral group are affected. The mesial are usually well preserved, showing abundance of large staining masses (Nissl).

In the dorsal segment a few motor cells show marked excess of pigment.

Most of the cells of Clarke's columns present axonal reaction of a severe grade.

In the medulla and pons there are marked changes of similar type, in the cells of almost all the cranial nuclei, except the hypoglossal. In the vagus nuclei from four to six cells in each section show stages of central chromatolysis.

Many cells in the descending root of the trigeminus, also the cochlear, and glosso-pharyngeal muscles, are affected.

Marked axonal reaction involving most of the cells in the facial nuclei. The cells of the inferior olives, and of the nuclei of Goll and Burdach present marked changes.

Cortex—The pia is quite generally thickened and shows a moderate cellular infiltration. Blood vessels of the cortex are considerably injected. In places a slight perivascular infiltration, chiefly of lymphoid cells. No marked changes of the walls.

Various areas of the cerebral cortex after fixation in alcohol were prepared by a modified Nissl method (paraffin sections). Throughout the cortex there is a general mild degree of so-called acute alteration of the cells. The pyramidal cells are swollen. The processes are visible farther than usual from the body of the cell.

The stainable substance is disintegrated into a pale, fine granular substance. In a few cells only, can one see the Nissl bodies at all and in these instances they have a fragmented appearance.

The nucleus is greatly swollen and the nuclear network quite distinct. The nucleolus often stains in a metachromatic way. In many nuclei there are coarse granules staining deeper than the nuclear network.

The Nissl bodies of the Betz cells are fairly well preserved. There are no marked glia changes.

With Marchi method there are found no notable degenerations in the cortex.

In the medulla considerable degeneration is seen in the restiform bodies, also a few fibers in the posterior longitudinal bundle, and the fillet are blackened.

Throughout the cord there is some marginal speckling, most marked in the direct cerebellar tract, and there are a number of blackened fibers in the middle of the posterior columns. Numerous fibers are affected in both the anterior and the posterior root bundles.

Summary of Anatomical Findings.—Slight arteriosclerosis. Hypostatic pneumonia. Fatty infiltration of the liver. Acute degenerations of many of the peripheral nerves. Axonal reaction in cells of the anterior horns; Clarke's columns and many cranial nerve nuclei. Degenerations in the posterior columns, direct cerebellar tracts, and the root bundles. Moderate "acute alteration" of the cortical cells.

CASE II. (FROM SERVICE OF DR. H. D. ARNOLD.)

H. B., travelling salesman, age about thirty-five. Family history negative. History of several slight attacks of rheumatism, and gonorrhea twice. Patient, at least for the last few years, has been an excessive drinker. Up to about a year ago he drank beer chiefly. He was persuaded to abstain entirely for about six months on account of chronic urethritis. Then he commenced to drink harder than before, taking several glasses of whiskey daily. Frequently was incapacitated for work. He began to have trouble with his feet in May, 1903. He complained of pain in the great toe of the right foot. This gradually extended to the other foot. His feet became swollen and he often cried with pain. Soon after the onset of this trouble his friends noticed a marked change in his disposition. He became very irritable and whimsical, and felt a personal affront when his opinion was crossed. He would often cry because of the supposed injury to his feelings. He became restless, quite secretive, and even suspicious. Formerly he had been quite open and communicative. It was noticed also that his memory was becoming poor. Many things of recent happening he was unable to recall. At one time he was consulted and agreed to a change of policy on the part of the company which he represented. A few weeks later when this change went into effect he was very bitter because he had not been consulted, denying all knowledge of the event. During the last six months he had frequently contracted bills and borrowed money, but later proved unable to recall the occurrence. This memory weakness increased until at the time of admission to the hospital he was unable to give a very satisfactory account of the last months of his life. His feet began to bother him so much in September that he finally consented to go to the hospital. He was admitted to the Boston City Hospital Sept. 29 1903.

His general condition was good. Muscular tremors were seen in hands and tongue. The pupils were normal. Heart and lungs were negative. The plantar surfaces of both feet were somewhat swollen, reddened and tender, particularly over the metatarso-phalangeal joints, which were extremely painful. The knee-jerks were lively. There was no atrophy or edema.

Oct. 3.—The condition of the feet had improved. The patient was fairly quiet, less tremulous, and sleeping about half the night. Temperature was normal.

Oct. 7.—Up and out of doors daily in a chair. Slight tremors. He slept six hours. There was less pain and swelling in the feet. Numbness in the right great toe.

Oct. 11.—Pain was more intense and tenderness more marked. No pains in the legs. General condition remained good.

Oct. 19.—Foot symptoms were the same. He complained of pains in the calves of his legs and was nervous at night. He was somewhat irrational and less thoroughly aroused. For several nights previous he had been getting out of bed and walking about the corridor, apparently not realizing, for the time being, his whereabouts. He slept about half the night.

Oct. 24.—He was irrational and delirious, required constant watching. Condition of feet seemed improved. He slept little. A day or two later became semi-comatose and unable to articulate and to swallow. There were no convulsions, no spasticity. Pupils reacted promptly to light. No paralysis, but incontinence of sphincters. Heart was rapid, irregular; sounds somewhat distant. No murmurs. Lungs showed occasional moist râles in the back. Knee-jerks were equal and normal. There was no Babinski reflex. He gradually became more and more somnolent, with increased difficulty in breathing. Death occurred Oct. 30, apparently due to failure of respiration.

Clinical Summary.—Acute alcoholic mental confusion following chronic neuritis, and of the type of Korsakoff's symptom-complex. Marked amnesia for recent events, irritability, increased emotional reaction, imperfect orientation and mild delirium.

Later developed difficulty in speaking and in swallowing. Death from vagus paralysis and failure of respiration.

Anatomical Findings.—(Autopsy by Drs. C. W. Keene and E. E. Southard).

Fairly developed, well-nourished white male of excellent musculature.

Peritoneal Cavity—Vessels of intestines are injected. No fluid. Many bubbles of gas show in mesentery of sigmoid flexure and descending colon. Culture shows bacillus aerogenes capsulatus.

Pericardial Cavity—Contains about 25 cc. straw-colored, serous fluid.

Pleural Cavities—Few fibrous adhesions lower left lobe to back and diaphragm.

Heart—Weight, 390 gms. Subepicardial fat greatly increased, envelops whole organ. On section, dark red color, rather firm, slightly granular in appearance. Fresh sections stained with Scharlach R., show very slight fatty change.

Few small, fairly firm, elevated, white patches in coronary arteries. One in anterior coronary covers vessel wall for 2.5 cm. from mouth.

Measurements—Tricuspid valve 12 cm.; pulmonary valve 7.5 cm.; mitral valve 10 cm.; aortic valve 7.5 cm.; left ventricle 2 cm.; right ventricle 0.6 cm.

Lungs—Fill cavities. Lower lobes dark red; upper shade to grayish pink showing black carbon mottling. Both crepitant throughout, but darker portions are slightly firmer to touch. On section dark parts drip blood; slight amount of muco-pus shows in bronchi.

Spleen—Weight, 145 gms. Very dark red, fairly firm, with rather loose capsule. On section, trabeculæ and Malpighian corpuscles clearly made out.

Liver—Weight, 2320 gms. Reddish brown color, on section red mottled and small drops.

Kidneys—Weight, 356 gms. On section dark red. Capsule strips easily leaving smooth surface. Cortex 6-7 mm. Pyramids distinctly marked. High blood content. Section stained with Scharlach R. shows slight fat.

Gastrointestinal Tract—Normal but for injected blood vessels.

Genital Organs—Right testicle threads out well. Left does not thread. Tubules cannot be made out and glandular tissue is replaced by opaque grayish, fibrous material, rather firm to touch, except at upper part of testicle.

Aorta—Shows many small, 2mm., elevated, fairly firm, yellowish white patches in ascending portion; several small ones around mouths of arteries in descending portion.

Head—Hair fairly thick and black. Scalp veins distended with fluid blood. Calvarium heavy, without diploe. Several deep excavations reaching to outer table, to receive groups of arachnoidal villi without dural sheaths.

Dura—Lax, nowhere adherent. Middle meningeal vein system injected. Sinuses filled with fluid blood, Arachnoidal villi slightly developed except at a few foci along longitudinal sinus.

Pia—Contains a moderate amount of clear fluid, especially at the vertex. The superficial cerebral venules are injected. Veins of sulci moderately injected. Vessels at the base surrounded by edematous fibrous tissue of normal depth. Circle of Willis normal.

Brain—Weight, 1510 gms. Substance pinkish gray. Consistence slightly softer over left vertex than elsewhere; the gyri here are somewhat wider than the gyri on the right, and are only roughly symmetrical with these.

Microscopical Findings.—Heart and lung—Not remarkable.

Spleen—Much congestion. Slight increase in reticulum. A few phagocytic endothelial cells. Occasionally vacuolated. Many eosinophiles and few polynuclear leucocytes. Much pigment. Arteries slightly thickened.

Liver—General vacuolation, which is most marked at the lobule centers. Foci of lymphoid and plasma cells with slight increase of periportal connective tissue. A few eosinophiles seen.

Kidney—Tubules in general are slightly dilated, contain granular material, and show flattened epithelium. The glomerular tufts are injected. Some tubules stain more intensely with eosin than others (hyaline). Very few vacuoles. The pyramids are injected and a few lymphoid and plasma cells are seen. Arteries very slightly thickened.

Testicle—Section shows some areas in which connective tissue increase has replaced tubules, but more generally there are areas of increased connective tissues containing atrophic tubules. Spermatogenesis sluggish in better preserved areas. Foci of cells of lymphoid type are seen. Arteries are thickened.

Nervous System.—Peripheral Nerves—The posterior tibial and external popliteal examined by Marchi method show numerous degenerated fibers in nerves of the right side. The nerves of the left side not so severely affected. Both vagi show the Marchi reaction of nearly one-half the fibers. No marked changes in optic nerves. Vessels show some intimal thickening. No cellular infiltration.

Cord—The anterior horn cells of the lumbo-sacral enlargement have acute changes, about one-third of the cells present early stages of the axonal reaction. There is slight swelling of the cell body and atrophy of dendrites with a varying degree of central chromatolysis and eccentric nucleus. One side seems to be more severely affected than the other. An occasional motor cell in the dorsal cord and one or two cells in sections from the cervical enlargement show a similar reaction.

In the cells of Clarke's column one may find two to four cells in a section, showing dislocated nuclei.

Nissl preparations from several layers of the medulla and pons present changes of the same type in cells of some of the cranial nerve nuclei. A large number of cells of the glossopharyngeal nuclei and the motor nuclei of the vagi show severe axonal reaction as well as many cells in the nuclei of the posterior columns.

Cerebral Cortex—Fibers of the pia are considerably separated by edema. In places a slight fibrous thickening and slight cellular infiltration. The vessels are all dilated and engorged.

Pieces from various parts of the brain after fixation in formalin were stained for general histological work, neuroglia and fat. The most marked changes were found in the frontal and paracentral convolutions.

In the paracentral convolutions there is a normal distribution of cells. The first glance at the specimen shows axonal reaction of the Betz cells. In about one-half there is a fine powdering of part of the stainable substance, affecting chiefly the center of the cell, but it may extend to the periphery especially the basal part. None of the cells is completely involved. The nucleus is frequently found displaced to the side or the upper extremity of the cell. There is a little pigment deposit in many of the cells. Many of the larger and medium-sized pyramids present similar alteration.

Occasionally there is found a large pyramid which is evidently necrotic. By the Nissl method the staining is very faint, the whole cell is much shrunken, and has irregular indistinct outline. The nuclear outline is not made out, but the nucleolus remains deeply stained.

In the layer of larger pyramidal cells there is a moderate increase in free nuclei in the ground substance. Many of the larger cells are beset with satellite cells. Frequently as many as six or seven may be counted around one cell, perhaps indenting it in one or two places.

A few of the glia cells in the neighborhood of the affected Betz cells are caught in mitosis. They are almost entirely of the earlier skein forms. One or two of aster type noted.

In the frontal cortex there are a few cells which show axonal degeneration. A few of the larger pyramids have a rarefied apex, and the nucleus is pushed to one side. Satellites are not so abundant.

There are no notable changes in the cerebellum.

Blood Vessels—There is a very marked general vascular engorgement of the brain and cord. A number of small microscopic hemorrhages are found, confined to the cortical gray matter of the frontal, parietal and temporal lobes. Occasionally two or three phagocytes engorged with pigment, together with free pigment, are seen.

The vessel walls do not present marked changes. In some the nuclei of the intima seem more prominent than usual, and there is an occasional area in the cortex with slight infiltration of lymphoid cells.

Sections prepared by the Marchi method show numerous degenerated fibers in the white matter of the motor cortex. Also many fibers in the frontal and temporal areas, but not so marked as in the paracentral region. The occipital lobes are comparatively free.

In the walls and around most of the larger vessels there is a large amount of degenerative product staining with osmic acid. In the medulla there is a slight speckling of the pyramidal tracts, also the fillet and posterior longitudinal bundles. More marked speckling is seen in the cerebellar afferent tracts.

Throughout the cord there is slight marginal speckling, more marked in the direct cerebellar tracts. The pyramidal tracts also show numerous blackened fibers. There is very slight degeneration in the posterior columns, except in the lumbo-sacral region, where there is quite intense degeneration, confined chiefly to the middle of one side. A few fibers in the anterior root bundles and numerous fibers in the posterior roots are affected.

Summary of Anatomical Findings.—General arteriosclerosis involving the aorta and coronaries. Fatty degeneration of the heart, liver, and kidneys. Acute bronchitis. Acute degenerations in the peripheral nerves of the lower extremities, and also in the vagi. Axonal reaction in cells of the anterior cornua, in Clarke's columns, some cranial nuclei, and the Betz cells of the cortex.

Vascular changes in the cord and cortex, with numerous microscopical hemorrhages throughout the cerebrum. Acute degeneration of the cortical radiations, and of both motor and sensory systems of the cord, as well as degenerations of the cord not easily reconcilable with the systemic changes.

In looking over the literature of Korsakoff's symptom-complex we find as a rule distinctly demonstrable changes in the brain of cases which have been studied anatomically. These changes are variable and not specific. In an analysis of these studies it is important to keep in mind the stage of the disease.

In an early stage changes are similar to those found in delirium. In most of the more chronic cases extensive degenerations (Marchi) have been found in the white substance of the cerebrum. Several authors found these degenerations more intense in the frontal, and especially in the central, convolutions.

In cases dying early in the disease these changes are often missed. Heilbronner reported two cases of fourteen days and six weeks respectively in which no demonstrable degenerations were found. The relation of the blood vessels to this condition is important. Marked congestion and prominence of the vessels have been very commonly seen. Few have found changes in the walls, Gudden has one case in which he found glass-like thickening of the walls.

Boedeker and Cole have reported cases with thickening of the vessel walls. A tendency to minute hemorrhage and blood extravasations in varying numbers has been frequently seen. These blood extravasations occur chiefly in the gray matter of the basal ganglia and around the third ventricle. Several authors mention the resemblance of some cases to polio-encephalitis.

Softening in the cerebellum was seen in one of Wernicke's cases. Gudden in two cases demonstrated foci of softening in the walls of the third ventricle, the basal ganglia and also the pons. Cramer and Wright also report cases.

Cortical cell changes are variable. "Axonal" reaction of Betz cells is described in cases of Faure, Wright, and Cole. In some of Bonhoeffer's cases there were changes in many of the cells, showing characteristics approaching those of Nissl's "acute alteration." In other cases there was evidently typical axonal reaction. Glia changes are found in most severe cases. Nearly all the cases of Korsakoff's psychosis reported show spinal cord changes. These are especially marked in the posterior columns and in the direct cerebellar tract, where there are ascending degenerations. In more severe cases degenerative changes are often found in the pyramidal tracts, and also degenerative changes, often more observed in the anterior and posterior roots. Heilbronner found that in the cord the affected areas agreed with the clinical and anatomical neuritis.

The two cases here studied show, upon an alcoholic and arteriosclerotic basis, acute degenerations of many peripheral nerves, axonal reaction in anterior horn cells, in cells of Clarke's column, and in some cranial nerve nuclei, and various non-systemic degenerations of intraspinal fibers, together with acute or axonal alterations of various cells of the cortex. In one case proliferating glia cells are found in the neighborhood of altered Betz cells of the motor cortex.

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